DISSERTATION

ON

A STUDY ON ACUTE SYMPTOMATIC SEIZURES

Submitted in partial fulfilment of

Requirements for

BRANCH - I D.M. NEUROLOGY

of

THE TAMILNADU DR.M.G.R. MEDICAL UNIVERSITY

CHENNAI

MADRAS MEDICAL COLLEGE

CHENNAI – 600 003.

AUGUST 2007

CERTIFICATE

This is to certify that this dissertation entitled **"A STUDY ON ACUTE SYMPTOMATIC SEIZURES"** submitted by **Dr. S. ARUNAN** appearing for **D.M.,** Degree examination in **August 2007** is a bonafide record of work done by him under my direct guidance and supervision in partial fulfillment of regulations of the Tamil Nadu Dr. M.G.R. Medical University, Chennai. I forward this to the Tamil Nadu Dr.M.G.R. Medical University, Chennai, Tamil Nadu, India.

HEAD OF THE DEPARTMENT,
Institute of Neurology,
Government General Hospital
Chennai – 600 003.

PROFESSOR OF NEUROLOGY
Institute of Neurology,
Madras Medical College
Government General Hospital
Chennai – 600 003

DEAN
Madras Medical College
Government General Hospital
Chennai – 600 003.

DECLARATION

I solemnly declare that the dissertation titled **"A STUDY ON ACUTE SYMPTOMATIC SEIZURES"** is done by me at Institute of Neurology, Madras Medical College & Govt. General Hospital,

Chennai, during 2005-2007 under the guidance and supervision of

Prof. V. NATARAJAN, M.D., D.M.,

The dissertation is submitted to The Tamilnadu Dr. M.G.R. Medical University towards the partial fulfillment of requirements for the award of **D.M., degree in Neurology.**

Place: Chennai

Date: 15/05/2007

Dr. S. ARUNAN
Postgraduate Student
D.M. in Neurology,
Institute of Neurology,
Madras Medical College
Chennai-600 003.

SPECIAL ACKNOWLEDGMENT

I gratefully acknowledge and sincerely thank **Prof. Dr. T.P. Kalanidhi Dean**, **Madras Medical College and Government General Hospital**, Chennai for allowing me to do this Dissertation and utilize the Institutional facilities.

ACKNOWLEDGEMENT

I would like to express my sincere gratitude to my beloved Professor and Head, Institute of Neurology, **Prof. Geetha Lakshmipathy** for her guidance and encouragement.

I profoundly thank my beloved Professors, **Prof. K. Muthuraj Prof. A. V. Srinivasan and Prof. R. M. Boopathy** for their guidance and encouragement.

This is one another fine moment to express my gratitude and indebtedness to my beloved **Prof. V. Natarajan** for his motivation, advice and valuable criticism, which enabled me to complete this work.

I am extremely thankful to Assistant Professors of Neurology **Dr. V. Kamaraj, Dr. C. Mutharasu, Dr. K. Bhanu Dr. S. Balasubramaniam and Dr. S. Velusamy** for their co-operation and guidance.

I am thankful to all my postgraduate colleagues for their constant support and sharp constructive criticism.

I am thankful to the Neurochemistry laboratory, Radiology Department and our EEG laboratory and all technicians for their cooperation.

I should thank each patient for the whole-hearted cooperation despite the morbidity they suffered.

ABBREVIATIONS AND ACRONYMS

- EEG : Electro Encephalo Gram.
- CSF : Cerebro Spinal Fluid.
- CT : Computerized Tomogram.
- MRI : Magnetic Resonance Imaging.
- ILAE : International League against Epilepsy
- GTCS : Generalized Tonic-Clonic Seizure
- CVT : Cerebral Venous thrombosis
- CVA : Cerebro Vascular Accidents.
- CP angle : Cerebello PontineAngle.
- AED : Antiepileptic Drug
- CTD : Connective Tissue Disorder

CONTENTS

INTRODUCTION

Seizures have been recognized since antiquity. One of the earliest descriptions[1] of generalized tonic-clonic seizure was recorded over 3000 years ago in Mesopotamia, which was attributed to the God of the Moon. Epileptic seizures were described in ancient cultures including those of China, Egypt, and India.

The word seizure is derived from Latin word "sacire", meaning, "to take possession of" indicating that the person having a seizure is possessed or at least out of control[2]. The clinical symptoms in seizures could be motor, sensory, autonomic, or psychic events although in practice, when a patient presents to a health care system with a seizure it is usually a convulsive (motor) seizure, either generalized or focal.

Acute symptomatic seizure can be defined as seizures that are provoked, situation related, occurring in close temporal relationship to a systemic or neurological insult directly or indirectly. Those affecting directly are due to denovo CNS disease like meningitis, encephalitis, stroke and head injury. Those affecting indirectly are systemic disorders leading to disordered homeostasis affecting the nervous system.

Cumulative observations of many clinical investigators, along with adjunctive neurophysiologic, imaging and genetic tools created a well-accepted diversity in the etiologies of seizures in various age groups. In adults and elderly the frequent causes are cerebrovascular disease, brain tumors, alcohol withdrawal, metabolic disorders, degenerative diseases and idiopathic[3].

With improved diagnostic facilities, the etiologies for acute symptomatic seizures can be identified and appropriately and effectively. This study is to analyze the acute symptomatic seizures occurring in adult age group.

REVIEW OF LITERATURE

Historical aspect [4]

Basic concepts surrounding seizures and epilepsy are found in ancient Indian medicine which dates back to Vedic period of 4500-1500BC. In the Ayurvedic literature of Charaka Samhita, seizures were identified and epilepsy was described as ***"apasmara"*** which means ***"loss of consciousness"***.

The foundation of our modern understanding of the derangement of function seen in seizures and epilepsy was laid in the 19th Century with the work of Hughlings Jackson. Working in Germany during the 1920s, Hans Berger, a psychiatrist, developed the human electroencephalograph. Another recent stimulus towards the understanding and treatment of seizures in the last few decades has been the development in neuroimaging. Such technology has revealed many of the more subtle brain lesions responsible for seizures.

Background:

Many diseases can cause paroxysmal clinical events. The correct diagnosis of the paroxysmal event is necessary to provide correct treatment. If the event is an epileptic seizure, the seizure type and associated clinical, electroencephalographic (EEG), and neuroimaging findings assist in determining the risk of seizure recurrence and the possible need to begin anticonvulsant therapy

Definitions Of Terms [10]

An **acute symptomatic seizure** is a seizure that occurs following a recent acute disorder such as a metabolic insult, toxic insult, CNS infection, stroke, brain trauma, cerebral hemorrhage, medication toxicity, alcohol withdrawal, or drug withdrawal.

A **nonepileptic event** is a clinical event presumed to be unrelated to abnormal and excessive neuronal discharge. An example of a nonepileptic event is syncope.

An **epileptic seizure** is a clinical event presumed to result from an abnormal and excessive neuronal discharge. The clinical symptoms are paroxysmal and may include impaired consciousness and motor, sensory, autonomic, or psychic events perceived by the subject or an observer.

An **unprovoked seizure** is a cryptogenic or a remote symptomatic seizure.

Frequency:

A first seizure is a sudden frightening event for the individual, onlookers and family members. Available data[5,6]. on an individual's lifetime risk of developing one episode of non-febrile seizures is atleast 4% [31]. A first seizure caused by an acute disturbance in brain function (acute symptomatic or provoked) is unlikely to recur (3 to 10 %). If a seizure is unprovoked, however, meta-analysis suggests that 30 to 50 % will recur; and after a second unprovoked seizure, 70 to 80 % recur, justifying the diagnosis of epilepsy [7,8,9] .

Race:

Racial differences have not been studied[5,6].

Sex :

Most authors report a small-to-moderate preponderance of men in their studies of first seizures in adults [18,20,28,29]

Age [3]

In practice it is useful to consider the etiologies of seizures based on age of the patient, as age is one of the most important factors determining both the incidence and likely causes of seizures.

Etiology of Acute Symptomatic seizures[3]

1. Medications
2. Alcohol related
3. Illecit drugs
4. Envioronmental toxins
5. Electrolyte Imbalance.
6. Glucose Disturbance
7. Organ Failure
8. Infection(CNS,Systemic)
9. Acute CNS disturbance.

10. Multi system diseases.
11. Hypoxic ischemic encephalopathy
12. Hypertensive Encephalopathy
13. Non Hypertensive Posterior leuco encephalopthy
14. Cancer associated
15. Organ transplantation

CLASSIFICATION OF SEIZURES

Seizures are divided into two broad categories--generalized and partial. Generalized seizures arise from both sides of the brain simultaneously. Partial (i.e., focal) seizures occur within one or more restricted regions of the brain and are a secondary effect of a localized physiologic or structural abnormality of the brain (e.g., tumor, dysplasia, stroke, trauma).

International League against Epilepsy Revised Classification of Epileptic Seizures[10]

1. Partial (focal, local) seizures:
    A. Simple - motor, somatosensory, autonomic, psychic
    B. Complex
        a. Impaired consciousness at outset
        b. Simple partial followed by impaired consciousness
    C. Partial seizures generalized tonic-clonic (GTC)
        a. Simple evolving to GTC

b. Complex to GTC

2. Generalized seizures (convulsive or non-convulsive)

    A. Absence seizures

    B. Myoclonic

    C. Clonic

    D. Tonic

    E. Tonic-clonic

    F. Atonic

    G. Combinations

3. Unclassified epileptic seizures

Evaluation of a first seizure [11]

The first step in evaluating a suspected seizure is to determine whether the event was, in fact, a seizure

Syncope is often mistaken for an epileptic seizure. During many syncopal episodes, clonic or myoclonic jerks occur in the distal portions of the extremities. Convulsive syncope occurs when there is severe or prolonged reduction of blood flow to the brain, resulting in an event resembling tonic-clonic seizure. Diagnosis is based on possible provocative factors in the medical history (e.g., pain, dehydration), physical examination (e.g., orthostatic blood pressure check), and studies such as electrocardiography and tilt table testing [12].

Another common imitator of epileptic seizure is the nonepileptic psychogenic seizure. No single feature reliably differentiates the two disorders. However, many ictal features of nonepileptic psychogenic seizure are uncommon in epileptic seizures [13.] For example, such features as gradual onset, stopping and restarting of motor activity, out-of-phase clonic movements of the extremities, vocalization in the middle of the seizure rather than at the start, pelvic thrusting, and lack of body rigidity are more common in psychogenic seizures than in tonic-clonic seizures In addition, the typical duration of a tonic-clonic seizure is 50 to 92 seconds, whereas the range for psychogenic seizures is 20 to 805 seconds[13].

Furthermore, some epileptic seizures have symptoms that are frequently misdiagnosed as psychogenic. Frontal lobe complex partial seizures often last less than 1 minute and sometimes include rocking, kicking, "bicycling," pelvic thrusting, genital manipulation, and cursing. Their lack of postictal symptoms also makes frontal lobe seizures difficult to differentiate from psychogenic seizures.

Other Nonepileptic Paroxysmal Events :

- Migraine (classic [with auras], basilar, confusional)
- Cerebrovascular event (transient ischemic attack)
- Periodic paralysis
- Sleep disorders (parasomnias, daytime amnesic episodes)

- Gastrointestinal disorders (reflux, motility disorders)

- Movement disorders (tics, Tourette's syndrome, nonepileptic myoclonus, paroxysmal choreoathetosis, shuddering attacks)

- Psychiatric disorders (panic, somatization, dissociation, conversion [nonepileptic psychogenic seizures])

- Drug toxicity and substance abuse

- Breath-holding spells

History of the event [11]

A description of the circumstances surrounding a paroxysmal event can provide important diagnostic clues. A witnessed, 90-second episode that involved loss of consciousness, stiffening, and jerking of the extremities followed by muscle soreness, headache, and the need to sleep for several hours afterwards strongly suggests a tonic-clonic seizure

KEY ELEMENTS IN HISTORY

Before the event

- Unusual stress (e.g., severe emotional trauma)

- Sleep deprivation

- Recent illness
- Unusual stimuli (e.g., flickering lights)
- Use of medications and drugs
- Activity immediately before event (e.g., change in posture, exercise)

During the event

- Symptoms at onset (e.g., aura)
- Temporal mode of onset: gradual versus sudden.

- Duration : brief (ictal phase <5 min) versus prolonged Stereotypy: duration and features of episodes nearly identical versus frequently changing

- Time of day: related to sleep or occurring on awakening

- Ability to talk and respond appropriately

- Ability to comprehend

- Ability to recall events during the seizure

- Abnormal movements of the eyes, mouth, face, head, arms, and legs

- Bowel or bladder incontinence

- Bodily injury

After the event

- Confusion

- Lethargy

- Abnormal speech

- Focal weakness or sensory loss (ie, Todd's paralysis)

- Headache, muscle soreness, or physical injury

Past medical history

A review of the events leading up to the seizure may reveal factors that suggest it was provoked. Causes of provoked seizures include alcohol withdrawal, substance abuse, hypoxia, fever, electrolyte imbalance, hypoglycemia, and sleep deprivation.

Drug history

Theophylline, meperidine hydrochloride , isoniazid , antipsychotic drugs (especially clozapine and phenothiazines), radiocontrast dyes, alkylating agents, and ß-lactam antibiotics are among the most commonly implicated medications in seizure.

However, many other drugs can cause seizure, including lidocaine hydrochloride, general anesthetics, tricyclic antidepressants , selective serotonin reuptake inhibitors, bupropion hydrochloride, acyclovir , ß-blockers, and decongestants (eg, phenylpropanolamine hydrochloride). Also, seizures can be provoked by alcohol withdrawal as well as use of cocaine.

Physical examination

A thorough physical examination can help uncover possible causes of a seizure. Findings may include evidence of trauma, infection, malignancy, congenital anomalies, and prior neurological events (e.g., focal weakness, plasticity suggesting previous stroke).

During an emergency department evaluation of a patient immediately after a seizure, vital signs should be measured and a general medical examination performed. Guidelines for physical examination are as follows:

- Examine the patient for injuries from the seizure or fall.
- Check oxygen saturation and auscultate the chest for possible aspiration.
- Measure heart rhythm and rate, blood pressure, and orthostatic changes for assessment of syncope.
- Auscultate for carotid murmurs or carotid bruits and sources of embolic stroke.
- Check for rapid pulses, which are often present after seizure and may help in evaluation of psychogenic seizures.

An electrocardiogram should be obtained to identify cardiac rhythm, detect possible ischemia, and measure the QT interval. Prolonged QT syndrome often presents with simple or convulsive syncope. Electrocardiography and 24-hour ambulatory continuous electrocardiographic (Holter) monitoring can help identify cardiac arrhythmias. The possibility of a recent myocardial infarction should be considered, particularly in elderly patients, in whom myocardial infarction may occur from the stress of a seizure.

Neurological examination

The purpose of the neurological examination is to identify focal or diffuse cerebral dysfunction. This information is particularly helpful in localization-related epilepsy. The presence of various features offers clues to the focus of a seizure. For example, aphasia suggests a left frontal, temporal, or parietal onset. Right or left hemi paresis suggests foci from the contra lateral motor cortex.

In initial evaluation of a seizure, patients should be observed for fluency of language, facial asymmetry, gaze preferences, and pupillary asymmetry. The last presents in patients who have herniation from brain swelling caused by parenchymal or epidural bleeding and in those who have a rapidly growing brain tumor. The presence of pronator drift may indicate subtle weakness not detected by strength testing. Sensory deficits suggest parietal lobe dysfunction. An extensor plantar response may be noted for some time after a seizure and is not necessarily a pathologic finding.

Diagnostic testing

Laboratory workup is an essential part of evaluation of seizure. Measurement of glucose, calcium, magnesium, thyroid hormone, and liver enzyme levels, as well as toxicology screening (including blood alcohol levels), may reveal common medical causes of seizures. A complete blood cell count may suggest infection, anemia, or sickle cell disease.

In patients suspected to have had an infection or a fever or to have exhibited abnormal behavior just before the event, lumbar puncture should be performed after assessment of the possible risks of the procedure (eg, coagulopathy, mass lesion). Patients who are immunocompromised because of corticosteroid use, recent transplantation, or HIV infection should undergo cerebrospinal fluid evaluation to detect possible fungal, bacterial, or viral infection. In patients with a systemic malignant condition, cytologic evaluation of cerebrospinal fluid can identify meningeal carcinoma.

Electroencephalogram:

- EEG should be performed within 24 hours of the seizure because it is significantly more sensitive when obtained during that period (King, 1998). If the routine EEG findings are normal, a sleep-deprived EEG should be performed.

- Standard EEG detects epileptiform discharges in 29% of patients. Standard EEG combined with sleep-deprived EEG shows epileptiform discharges in 48% of patients (van Donselaar, 1992)[20].

- In 2000, Simpson et al described a case in which the placement of an insertable loop recorder, an important new tool in the diagnostic evaluation of patients with syncope, led to an unexpected diagnosis of a seizure. Whenever cardiovascular causes are considered as the cause of a patient's spells but cannot be proven with conventional investigations, the use of the insertable loop recorder should be considered.

- Schreiner and Pohlman-Eden studied the value of an EEG taken within 48 hours of the first

seizure in an adult. They found that 38.0% of patients without seizure recurrence had normal EEGs, while only 10.2% of patients with seizure recurrence had normal EEGs. Focal epileptiform activities were found significantly more frequently (26.5% vs. 13.0%) in patients with seizure recurrence than in patients without seizure recurrence.

Limitation of EEG[30]:

An estimated 0.4% of adults and 2.8% of children who have never had a seizure may have interictal epileptiform discharges. Furthermore, a normal EEG does not refute the diagnosis of epilepsy. The initial EEG reveals epileptiform activity in only 40% of the patients with seizures

Imaging studies

The role of imaging studies depends on the stage of evaluation. Immediately after a seizure, computed tomography can detect the presence of bleeding or gross structural lesions. However, magnetic resonance imaging is the study of choice because it is more sensitive and specific for evaluating structural lesions and brain parenchyma. Particular attention should be directed to the hippocampus for evaluation of lesions (e.g., mesial temporal sclerosis) and to the cortical architecture for detection of abnormalities (e.g., dysplasia).

Are Antiepileptic drugs needed after a first seizure? [14]

Drug treatment after first seizure is controversial. Too large recent randomized studies of children and adults compared antiepileptic drugs with, no treatment after a first seizure and came to an identical conclusion. Any decision to start treatment must weigh the risk of another seizure against the risks of side effects from chronic drug treatment.

Risk factors for recurrent seizures include the following[15]

- Age younger than 16 years: Musicco et al found that children younger than 16 years had almost double the risk of recurrent seizures as adolescents and adults aged 16-60 years [16]Remote symptomatic seizure (Annegers, 1986; Hauser, 1990; Berg[17], 1991): In the case of seizures after a first stroke, Labovitz et al found that lesion location and stroke subtype are strong predictors of early seizure risk, and early seizures are a predictor of recurrent seizures (Labovitz[31], 2001).

- Seizures occurring between midnight and 8:59 am [18]

- Prior provoked seizures[19]

- Remote symptomatic seizure in a patient whose sibling is affected with epilepsy [19]

- Status epilepticus or multiple seizures within 24 hours as the initial remote symptomatic seizure [19]

- Partial seizures

- Todd paralysis in patients with a remote symptomatic seizure

- History of neurological deficit from birth such as cerebral palsy or mental retardation [17]

- Abnormal examination findings in patients without a remote symptomatic seizure [26]

- CT scan that shows a brain tumor [18]

- EEG that shows epileptiform discharges

  - In patients with a first seizure and no known etiology, van Donselaar obtained a routine EEG in all cases and a second sleep-deprived EEG if the first EEG did not show epileptiform discharges. His pooled results showed the following 2-year cumulative risks of seizure recurrence: in patients with epileptiform discharges, 83%; in patients with nonepileptiform abnormalities, 41%; and in patients with normal EEGs, 12% [20].

  - In 1997, Beghi et al[22] found that epileptiform discharges were associated with a 1.5- to 3-fold increase in the risk of seizure recurrence.

  - In 1993, Musicco et al[16] found that epileptiform discharges were associated with a 1.7-fold increased seizure recurrence risk.

  - Berg and Shinnar found that epileptiform discharges were associated with a 2-fold increased seizure recurrence risk [21].

- In 1990, Hauser et al[19] found that generalized spike and wave increased the risk of recurrent seizure in patients with no known etiology.

- In 1997, Beghi et al[22] found that an abnormal EEG finding and the presence of an underlying etiology (remote symptomatic) are the most consistent predictors of recurrence.

If drug treatment is considered, which drug is preferred?

If drug treatment is considered after first seizure, the chosen antiepileptic drug should have high efficacy, good tolerability and low interaction potential and allow a good quality of life, especially since half of all patients would never have another seizure without treatment. The starting dose should be in the lower range

If an underlying epilepsy syndrome has been established, the following antiepileptic drugs are available[3].

	GTCS	**Partial**	**Absence**	**Myoclonic,**
First Line	Valproic Acid Lamotrigine	Carbamazepine Phenytoin	Valproic Acid Ethosuximide	Valproic Acid
Alter-natives	Phenytoin Carbamazepine	Topiramate Levetiracetam	Lamotrigine Clonazepam	Lamotigine Topiramate

Further Inpatient Care:

- Many patients who have a seizure recover spontaneously and fully with normal consciousness after a short time interval. Patients with incomplete recovery or a prolonged postictal state may require inpatient hospitalization[23].
- Inpatient management may be necessary if the clinical course is complicated by other medical problems requiring inpatient management.

A short hospitalization may be necessary for patients who are at risk of recurrent seizures and have no adequate supervision at home. Patients admitted from an emergency department had a 16.8% risk of an early recurrent seizure during their brief hospitalization[24]. This risk of early recurrent seizures was higher than reported in other studies [16,17,19]

Summary

The first step in evaluation of a presumed seizure is to determine whether the event was indeed a seizure and which diagnostic studies are needed. The second step is to correctly diagnose the seizure on the basis of the medical history and findings from the physical, neurological, and laboratory evaluation. The third step is to decide whether drug treatment is necessary. Every paroxysmal event is unique, and not every seizure needs to be treated. When treatment is deemed appropriate, an antiepileptic drug should be chosen after consideration of the risk-benefit profile of the available agents.

AIM OF THE STUDY

- To study the etiologic profiles of acute symptomatic seizures in adult patients aged more than 18 years of age.

- To analyze the age / sex distribution, presenting history, clinical findings and investigations at admission in the study group.

MATERIALS AND METHODS

The study was done in the setting of the Institute of Neurology Government General Hospital, Chennai. The study had collaborations with the Institutes of Internal Medicine, Biochemistry, Pathology, Radiology and Microbiology.

The study was observational in nature designed to analyze patients in age group more than 18 years of age and who presented with first onset seizures. The sample size was 150 and the study period was from July 2005 to March 2007.

INCLUSION CRITERIA

Cerebrovascular insults : Seizure occurring within 7days of an acute occlusive or hemorrhagic insult or in association with extension or progression of the primary insult.

CNS infection : Seizures occurring during the course of an active CNS infection. Evidence of infection at the time of the seizure from laboratory tests or clinical symptoms was required.

CNS neoplasm : Seizures occurring as presenting symptoms of a primary or secondary neoplasm, or seizures occurring in the immediate postoperative period were considered acute symptomatic seizures.

Toxic : Seizures occurring at the time of exposure to a systemic toxin or a neurotoxin. This exposure may have been related to the use of prescription drugs (e.g., aminophylline),

recreational drugs(e.g., cocaine), associated with patient-initiated drug overdose (e.g., imipramine), or related to environment exposures (e.g., carbon monoxide,organophosphates, or camphor).

Metabolic : Seizures occurring at the time of systemic dysfunction manifestedby systemic disturbances such as electrolyte imbalance, uremia, hypoglycemia,or cerebral anoxia.

Withdrawal : Seizure occurring after abrupt discontinuation of substances such as alcohol or barbiturates in individuals presumed to be habituated to their use.

Seizures associated with **acute drug intake** were classified as toxic. **Eclampsia Peripartum seizures** associated with other symptoms of eclampsia such as proteinuria and hypertension.

Encephalopathic : Seizures attributed to insults at the time of or after acute, deprivation generally associated with global perfusion failure(cardiac arrest) or selective deprivation (respiratory arrest).

EXCLUSION CRITERIA

- **History of trauma**

Clinical data was collected from patients and witnesses in a systematic manner and added to a database, which included a checklist of seizure antecedents and the symptoms associated with seizure.

The first task was to ascertain if at all, the presenting complaint is a seizure. In a few instances, even when the presenting history was ambiguous seizure recurrences were witnessed for confirmation. The clinical diagnosis on the seizure type, whether partial or generalized was made.

In-depth probes in the history for provocation factors and features suggesting organicity were attempted. Significant past medical history if any were noted. A thorough clinical examination was performed at the time of admission and relevant findings recorded. A routine metabolic screening, which included blood sugar, urea, serum creatinine, electrolytes and liver function tests (if indicated), were done at the time of admission.

Lumbar puncture and CSF analysis was done if infective etiologies were suspected.

Earliest possible EEG was attempted and was performed using 32 channel digital EEG recorder.

CT brain plain study in all patients and contrast studies when necessary were done in all patients in the study group. MRI brain was done when indicated.

Limitations were encountered in affordability of patients for MRI scanning. Early EEG (within 24hours of onset of seizures) could not be performed due to delay in referral of the patients to this institution and because of the time taken for stabilizing patients. EEG could not be done in a some cases owing to emergency surgical interventions

OBSERVATION

Seizures in 150 patients in the age group of 18 to 82 are studied; of which 88are males and 62 are females.

Table 1

SEX DISTRIBUTION OF PATIENTS (n=150)

In this study the number of males was more than females in the ratio males : females 1.4:1.

TABLE 2

SHOWS THE DISTRIBUTION OF VARIOUS AGE GROUPS IN THE STUDY

Males (n=88)

Demographic profile of male patients shows maximum frequency was in the age group 61-70 years closely followed by 31-40years and 41-50 years.

Females (n=62)

Demographic profile of male patients shows maximum number of cases in the age group 51-60 years ,closely followed by 61-70years and 21-30 years.

Total(n=150)

Of the 150 patients maximum number of cases were in the age 61-70 years age group followed by age group 51-60 years. The frequency was least with cases in 1st decade and 8th decade.

TABLE 3

SEIZURE TYPES FOUND IN THE STUDY : (n=150)

The seizures are grouped as per international league against epilepsy-revised classification of epileptic seizures as partial seizures and generalized seizures. A partial seizure with secondary generalization was found in 10% of the study group.

TABLE 4

COEXISTENT NON CONVULSIVE SYMPTOMS AT ADMISSION

Limb weakness was noted in 68% of patients at the onset and it was the nonconvulsive symptom seen maximum in the study.43%had headache, 25% had fever whereas vomiting and confused state was seen in 21% each of the cases. Visual disturbances was seen in 15% cases.

TABLE 5

OBSERVATION IN HISTORY FOR ORGANICITY OF SEIZURES

Organicity of the seizures was assessed by analyzing the symptoms elicited by detailed history taking. Postictal confusion was present in 63%patient,frothing from mouth in 38%,Seizures during sleep in 21% and bladder and bowel incontinence in 19% cases. Tongue biting and bodily injuries were each present in 4% cases.

TABLE 6

ENUMERATES THE PROFILES OF SIGNIFICANT PAST MEDICAL HISTORY OBSERVED IN THIS STUDY.

Past medical history	No of patients
Diabetes	02
Hypertension	06
Both diabetes and hypertension	14
Renal failure	03
Connective tissue Disorder	05
Pulmonary tuberculosis	09
Heart disease	04

Past medical history showed 14 patients to be having diabetes and hypertension, diabetes alone in two and hypertension in six. Pulmonary tuberculosis was present in nine ,and connective tissue disorder in five.

During an emergency evaluation of patients at admission vital signs were monitored which revealed 16% of patients were hemodynamically unstable. Neurological examination revealed abnormality in 65% of patients.

TABLE 7

SPECTRUM OF NEUROLOGICAL SIGNS AT ADMISSION

Motor system abnormalities were present in 48% patients ,39% had altered sensorium ,19% had signs of meningeal irritation ,13% had cranial nerve abnormalities and cerebellar signs were present in two % patients.

TABLE 8

METABOLIC ABNORMALITIES IN PATIENTS AT ADMISSION

Metabolic abnormalities at the time of admission were investigated, as they are among the most readily treatable causes of seizures. The abnormalities in metabolic parameters were noted in 40 patients in this study hyponatremia and hypoglycemia was present in 13 patients each.

TABLE 9

EEG in the study

EEG was taken after stabilizing the patient and all were taken in the interictal period. EEG was done in 117 of the 150patients (78%) in this study. Abnormalities were found in 69 of the 117 patients subjected to EEG

The most common observed pattern in EEG was a diffuse slow wave pattern during the interictal period.**TABLE 10**

CT ABNORMALITIES IN THE STUDY GROUP

Cortical atrophy was found in combinations with various other findings Radiologist opinion was obtained on all CT scans. CT scan brain was done in all the patients in the study group. MRI scanning of brain was done only in 34 patients of the study group. MRI was done

only in necessary cases when CT was not fully contributory. Besides improvement in details of CT findings MRI was helpful in uncovering lesions missed in CT.

TABLE 11

NEW LESIONS UNCOVERED IN MRI

MRI brain revealed new lesions in 16 patients. Five each infarcts and granulomas were deducted in patients in whom CT brain was normal. Two each cases of CVT and encephalitis and one each of tumour and AV malformation were detected anew by MRI brain.

TABLE 12
ETIOLOGY PROFILES IN THE STUDY GROUP WITH MEAN AGE (n=150)

Others include, Tumour + Metabolic-1, CVT + Metabolic 1, CTD + Metabolic -1, Toxic 1, TIA -1, Drug Induced -2, Inf+CVA 1

Five of the 150 patients presented with status epilepticus.(one CVT, two Eclampsias and two hypoxic encephalopthies)

At least one recurrence in the month following first seizure was noted in 61 (41%) patients. In hospital death occurred in two patients who were admitted for first seizures,One eclampsia patient and another hypoxic encephalopathy following coronary artery disease .

DISCUSSION

The study group comprised of 58.67% males and41.33% females. Most authors report a small-to-moderate preponderance of men in their studies of first seizures in adults (van Donselaar[20], 1992; Musicco[28], 1997; Hopkins[18], 1988; King[29], 1998). A male to female ratio of **1.4: 1** is observed in this study, a trend noted in other studies.

Analyzing the age groups in this study the maximum incidence of first onset of seizures is found in the age interval of 61 to 70 years. Studies have shown that incidence of new onset seizures above age 65 is even higher than first year of life – 135 per 100000 vs. 79 per 100000 . In females the incidence was higher in 51-60 age group and equal to that of 61-70 in 21-30 years age group. The mean age for most of the common etiologies in this study was 51-60. The less common etiologies in this study, in seizures due to infective etiology, CVT, Hypoxic encephalopthy & AVMs, seizures occurred at mean age of 31-40 years. In eclampsia and CTD patients seizures occurred at the earliest age (25 years)in this study.

The seizure type classified in this study as per International League Against Epilepsy-revised classification of epileptic seizures revealed generalized seizure in 68.67% and partial seizure in 31.33%.

Zhu PG[33] studied new onset seizures in the ages between 20 and 80 revealed generalized seizures in 64% and partial in 30%.

Retrospective study of Perez et al in 250 patients with late onset seizures revealed 59% generalized and 41% partial in nature.

The observation of seizure types in this study is almost similar to the above-mentioned studies.

In contrary, a recent study of Perez-*Lopez* [34] *identified* partial seizures as the most common seizure type in adults

TABLE 13

COMPARES SEIZURE TYPE ENCOUNTERED IN THIS STUDY WITH VARIOUS STUDIES.

The seizure typing in this study was entirely made with history. The grey area in relying on history in classification of seizures is in the fact that the focal onset of a seizure is often missed and witnesses' attention is often drawn to the person only after an event becomes generalized.

Limb weakness and headache were the most common non-convulsive symptom, which the patients / attenders complained at admission. Fever presented at admission in 25% of patients in this study. It is important here to reemphasize that fever is one of the provocating factors for seizures. Vomiting presented in 90% at the time of seizures in patients with tumors. Pappiloedema was present in 20% of patients with tumors.

Postictal confusion was the most frequent factor present in the history to suggest organicity. Seizures during sleep occurred in 35% of patients with tumors and 20% with CVA and in all patients with granuloma.

A history of alcohol intake, in most of the days of a week for more than ten years was present in 20% of patients.

Previous history of diabetes was present in 16 patients, two of whom presented with hypoglycemic seizures. Both of them were on glibenclamide and one of them was found to have renal failure. One patient presented with seizures associated with a nonketotic hyper osmolar state.

Motor system abnormalities and altered sensorium were the most common neurological signs present at the time of admission. It was the motor system abnormality in clinical examination, which most frequently predicted an abnormality in the CT scan.

Of the five patients admitted with status epilepticus two each were found to have Eclampsia and hypoxic encephalopthy and one had CVT. Signs of meningeal irritation were present in four patients with tumor etiologies and three patients with CVA besides presenting in all patients with meningitis. Sixth and seventh were cranial nerves comprised most of the cranial nerve abnormalities. In one patient with CP angle tumor multiple cranial nerve palsies were noted. The patient who presented with cerebellar signs was later found to have posterior circulation stroke

Infective etiology accounted for 14.67%,which formed the second largest group in this study. Of the 22 patients 10 had tuberculous meningitis, six had pyogenic meningitis and six had encephalitis(two had HSV encephalitis)

Metabolic abnormalities contributed to etiology in 13% of patients and most of them were readily treatable, hence a thorough search for these factors should be the early priorities. The most common metabolic abnormality was hyponatremia which was often encountered as an associated finding with other etiologies.

Two patients had drug induced seizures ,one developed seizures after intravenous lignocaine when he was admitted for the treatment of myocardial infarction and another after flourescin angiography done in ophthalmic hospital.

The study newly detected eight patients to have Type 2 diabetes, 11 to have hypertension and four to have both. Renal failure was detected in three of the patients in the group.

EEG was done in 117(78%) of the 150patients in the study. Abnormalities were found in 69(59%) of the EEG's done. The average period from the onset of seizure to the record of EEG was six days, owing to the late referral of patients to this institution and to the time taken to stabilize the patient before shifting to EEG room. The yield of abnormalities in the EEG in this study could have been better if it were done more early or special methods such as continuous EEGs and sleep deprived EEGs were adopted[20].

The most common abnormality in EEG was diffuse slowing of background activity. Anti-convulsant drugs slow the normal background rhythm in EEG[3] and almost 80% of the patients in the study group were under the anti convulsant drugs when EEG was performed, which explains the predominance of diffuse slowing pattern in the EEG.

When the other investigations were inconclusive, “focal findings in the EEG originating from the temporal lobes” were recorded in two patients, which helped in the diagnosis of encephalitis.

CT scan was done in all patients in the study group, in which the abnormalities contributed to the etiologies in 42% of patients. Cerebral atrophy (41%) was the most common abnormality present in the scan report but had no relevance with any etiology.

Abnormal CT findings in this study included infarct (23.33%), tumors (12%), parenchymal hemorrhage (06%) and ring enhancing lesions (03.33%)

CT findings in the study of new onset seizures by Sayette V[34] et al found cerebral atrophy in 29%, CVA in 75%, tumors in 5%.The spectrum of CT findings differs from this study in that cortical atrophy is more, CVA almost the same and tumours are also more in this study.

In the study of Zhu PG[32], CT scan findings were compatible with CVA in 16% , tumors in 13% , atrophy in 7% and trauma in 8%. This study was done in age group ranging from 20 to 87 years. In the present study atrophy was present in 41%,CVA in 30%and tumours in 12% patients.

Affordability was a limiting factor for MRI studies in the study group. MRI despite improving the descriptions of lesions already studied in CT scans was instrumental in uncovering new lesions in 16 of the patients in the study, in whom all other investigations were otherwise normal. The new lesions uncovered were five each with granulomas and infarcts, two each with encephalitis and CVT and one each with tumour, infection and AVM.

Etiologic profiles revealed CVA, tumors, metabolic causes, CNS infections and alcohol withdrawal seizures contributing to 68% in this study. CVA was the single most common etiology uncovered in this study.

In the Minnesota study, the most prevalent underlying condition accounting for seizures in the elderly was stroke. Of the patients in the CVA group, 28 had infarcts and five had hemorrhages. Twenty eight patients presented with acute symptomatic seizures at the onset of the stroke..

Lesser and coworkers suggested that the acute and delayed post stroke seizures have different mechanism, the former related to “transient cytotoxic metabolic alterations” and latter to structural changes, especially extravasations of blood and deposition of iron.

Tumors contributed to 12.67% of etiologies in this study. Of the 19 patients with tumors, 16 had primary CNS tumor against three patients diagnosed to have secondaries. Tumor as etiology in various studies are as follows, Montréal neuro institute 36% (study age over 50),mayo clinic 22% (study age over 60), Liege Belgium study (age 55 to 64) 21%, Glasgow Scotland 12% (study age - elderly) and Denver general hospital 1% (study age over 69).

Tumor as etiology in adult patients lies between 1% and 36% in various studies and the result of this study (12.67%) lies somewhere in the midpoint of this spectrum

Hyponatremia, hypoglycemia, hyperglycemia and renal failure contributed to 13% of seizures in the study. They were the most readily treatable causes, especially those patients detected to have hypoglycemic seizures. Hence a review at the metabolic parameters at admission is mandatory and when detected is most rewarding for the treating physician.

History was the tell tale evidence in patients diagnosed to have alcohol related seizures which formed 10% of etiologies. Of the 14alcohol related seizures 12 had withdrawal seizures and two had alcohol excessive intake causing seizures. Alcohol related seizures were present in 2 females .

Granulomatous etiology for first onset seizure was found in 06.67 percent of the study group. Of the ten patients six had neurocysticercosis and 4 had tuberculomas in brain.

Seizure was associated with CTD 1 % patients all of whom were young females with mean age of 25 years. Infective etiology accounted for 14.67%,which formed the second largest group in this study. Of them 10 had tuberculous meningitis ,six had pyogenic meningitis and six had viral encephalitis (two had herpes Simplex infection)

TABLE 14

COMPARES ETIOLOGIC PROFILES OF THIS STUDY WITH THAT OF MINNESOTA STUDY

ETIOLOGIES	THIS STUDY (IN PERCENTAGE)	MINNESOTA STUDY (IN PERCENTAGE)
CVA	18.67	6.5
Infection	14.67	5.5
Metabolic	12.67	3.5
Tumours	12.67	2.9
Alcohol Related	9.33	6.2
Eclampsia	3.33	0.5

.

The various etiologies of the Minnesota study[5] is very much different from that of this study probably that study involved patients from all age groups including new born.

In a study in Hyderabad[37] CVA formed 14% of causes for acute symptomatic seizures in adults and CVT forms one third of those patients in younger age group. In that study Single CT enhancing lesions and CNS infections formed almost 77% of the cases.

RESULTS

- The mean age of patients in the commonly encountered etiologies, in the study was around fifty to sixty years.

- The mean age of CTD as etiology, was the least in this study.

- **Generalized seizures (68.67%) were the most common seizure** type encountered in the study.

- Limb weakness and headache were among the most common non-convulsive presenting symptom.

- In the clinical examination, motor system abnormality was the most consistent factor that predicted an abnormal CT scan.

- EEG, which was done in 78% of the patients in the study recorded abnormalities in 59%.

- **Cerebrovascular accidents were the most frequent etiology for the first onset seizure in adults in this study**.

- Literature reveals a great diversity in the proportions of tumors forming etiology of seizures in later ages (1% to 36%). This study established **tumors as etiology in 12.67% of patients**.

- CVA, CNS infections, tumours, metabolic causes and alcohol withdrawal formed 75% of the etiology of seizures.

- In this study, 8%(twelve patients) mandated neuro-surgical intervention.

- Metabolic abnormalities contributed to etiology in 12.67% of patients.

- CT detected abnormal lesions in 42% of cases.

- **MRI was instrumental in uncovering new lesions in 16 patients.**

CONCLUSIONS

In a patient with new onset seizures more than 18 years, all efforts to identify the etiology should be made

- Given the age of patients more than eighteen years with a seizure does not exceedingly favor any specific etiology.
- Thorough search to rule out metabolic factors and infective causes as cause for seizures should be an early priority as these conditions are treatable.
- CT brain and MRI are indispensable in patients more than 18 years with new onset seizures.

REFERENCES

- Goldensohn ES, Porter RJ, Schwartzkroin PA: The American Epilepsy Society: an historic perspective on 50 years of advances in research. Epilepsia 1997 Jan; 38(1): 124-50
- Neurology in Clinical Practice edition, 4th Edition By Walter G. Bradley, DM, FRCP
- Seizures–Medical Causes and Management,NormanDelanty , Humana Press ,Totowa ,New Jersey 1st Edition-2002
- WHO fact sheet N º168 revised February 2001
- Hauser WA, Annegers JF, Kurland LT . Incidence of epilepsy and unprovoked seizures in Rochester, Minnesota; 1935 – 1984. Epilepsia 1993; 34:453- 68
- Foregren L, Bucht G, Eriksson S, Berg mark L. Incidence and clinical characterization of unprovoked seizures in adults: a prospective population-based study. Epilepsia 1996; 37: 224-29.
- Camfield PR, camfield CS ,Dooley JM ,Tibbles J, fung T, garner B. epilpeasy after a first unprovoked childwood. Neurology 1985;35:1657-60.
- Hauser WA, rich SS, lee JR annegers Jf ,Anderson VE. Risk of recurrent seizures after two unprovoked seizures. N Engl J med 1998;338:429-34.
- Shinnar S, berg AT, O'Dell newstein D, Moshe SL , hauser WA.predictors of

multiple , seizures in a cohort of children prospectively followed from the time of their first unprovoked seizures. Ann Neurol 2000;48:140-7.

- ILAE Epilepsia 1989, 30, 389-399
- Vol 111 / No 1 / January 2002 / Postgraduate Medicine
- Kochiadakis GE, Rombola AT, Kanoupakis EM, et al. Assessment of autonomic function at rest and during tilt testing in patients with vasovagal syncope. Am Heart J 1997;134(3):459-66
- Gates JR, Ramani V, Whalen S, et al. Ictal characteristics of pseudoseizures. Arch Neurol 1985;42(12):1183.
- BMJ vol 332 11 feb 2006
- Author: William J Nowack, MD, Associate Professor, Department of Neurology, University of Kansas Medical Center e MEDICINE SPECIALITIES
- Musicco M, Beghi E, Bordo B: Randomized clinical trial on the efficacy of antiepileptic drugs in reducing the risk of relapse after a first unprovoked tonic-clonic seizure. First Seizure Trial Group (FIR.S.T. Group). Neurology 1993 Mar; 43(3 Pt 1): 478-83.
- Annegers JF, Shirts SB, Hauser WA, Kurland LT: Risk of recurrence after an initial unprovoked seizure. Epilepsia 1986 Jan-Feb; 27(1): 43-50.

- Hopkins A, Garman A, Clarke C: The first seizure in adult life. Value of clinical features, electroencephalography, and computerised tomographic scanning in prediction of seizure recurrence. Lancet 1988 Apr 2; 1(8588): 721-6
- Hauser WA, Rich SS, Annegers JF, Anderson VE: Seizure recurrence after a 1st unprovoked seizure: an extended follow-up. Neurology 1990 Aug; 40(8): 1163-70.
- Van Donselaar CA, Schimsheimer RJ, Geerts AT, Declerck AC: Value of the electroencephalogram in adult patients with untreated idiopathic first seizures. Arch Neurol 1992 Mar; 49(3): 231-7
- Berg AT, Shinnar S: The risk of seizure recurrence following a first unprovoked seizure: a quantitative review. Neurology 1991 Jul; 41(7): 965-72
- Beghi E, Berg AT, Hauser WA: Treatment of Single Seizures. In: Engel J Jr, Pedley TA, eds. Epilepsy: A Comprehensive Textbook. Philadelphia, Pa: Lippincott-Raven; 1997: 1287-1294.
- Moore-Sledge CM: Evaluation and management of first seizures in adults. Am Fam Physician 1997 Sep 15; 56(4): 1113-20
- Tardy B, Lafond P, Convers P, et al: Adult first generalized seizure: etiology, biological tests, EEG, CT scan, in an ED. Am J Emerg Med 1995 Jan; 13(1): 1-5
- Bora I, Seckin B, Zarifoglu M, et al: Risk of recurrence after first unprovoked tonic-clonic seizure in adults. J Neurol 1995 Feb; 242(3): 157-63

- Camfield PR, Camfield CS, Dooley JM, et al: Epilepsy after a first unprovoked seizure in childhood. Neurology 1985 Nov; 35(11): 1657-60
- Chadwick D, Smith D: Epileptology of the first-seizure presentation. Lancet 1998 Dec 5; 352(9143): 1855; author reply 1856
- Musicco M, Beghi E, Solari A, Viani F: Treatment of first tonic-clonic seizure does not improve the prognosis of epilepsy. First Seizure Trial Group (FIRST Group).Neurology 1997Oct; 49(4): 991-8
- King MA, Newton MR, Jackson GD, et al: Epileptology of the first-seizure presentation: a clinical, electroencephalographic, and magnetic resonance imaging study of 300 consecutive patients. Lancet 1998 Sep 26; 352
- Walczak TS, Jayakar P. Interictal EEG. In: Engel J Jr, Pedley TA. Epilepsy: a comprehensive textbook. Philadelphia: Lippincott-Raven, 1997:831-48
- The Lancet . vol 352 . sep 26,1998
- Zhu PG Neurology Department, Tong Ji Hospital, Tong Ji Medical university, Wuhan. PMID;2282883.
- Perez lopez JL et al Acta Neurol Scand. 1985 Oct:72(4);380-4
- de la Sayette V et al Canadian Journal of neurosciences Aug 1987;14(3);286-9.
- seizure disorders; the changes with age. Epilepsia 33 (suppl 4);S6-S14,

1992.

- Chandra B: First seizure in adults: to treat or not to treat. Clin Neurol Neurosurg 1992; 94 Suppl: S61-3.

- Acute symptomatic seizures - incidence and etiological spectrum: a hospital-based study from South India. Murthy JM, Yangala R. Seizure. 1999 May;8(3):162-5

PROFORMA FOR EVALUATION

Name : **Age :** **Sex :**

Address :

Occupation : **Contact No.:**

Medical unit Prof **OP/IP no** **Ward** **DOA** **DOD**

Neuro Unit Prof **Min no** **EEG no**

Referred from

Elaboration of seizure

Witness of seizure **Reliability**

Before the episode

Recent illness (headache / fever)

Unusual stress

Medications

Last alcohol intake

Last meal

Sleep deprivation

Activity just before seizure

During the episode

Time of day

Aura

Duration

Ability to talk & comprehend

Ability to recall events

Movements of eyes face arms legs

Tongue bite frothing

Bowel / bladder incontinence

Bodily injuries sustained

After event

Confusion duration

Focal neurological deficits

Headache

Any other significant symptoms

SIGNIFICANT PAST HISTORY

Diabetic : yes / no duration & treatment

Hypertension CAD CKD

tuberculosis

any others

alcohol intake y / n duration freq quantity

last intake

smoking

family h/o seizures

<u>Clinical Examination</u>

General exam

Neuro cut markers

Vitals : BP Pulse RR Temp

CNS :

at presentation Time after seizure

signs of meningial irritation

higher functions cranial nerves

motor system sensory system

cerebellum

CVS : RS: P/A :

COURSE DURING HOSPITAL STAY

INVESTIGATIONS :

Hematology TC : DC: P L E B HB : ESR:

Biochemistry sugar urea creatinine Na k Ca

CSF analysis

Others : **ECG :**

Cxr : **ECHO :**

CT BRAIN : MRI BRAIN :

EEG :

Treatment

MASTER CHART

Sl.No	Age	Sex	SZT	HOR	SPH	HAD	NEA	EEG	CTS	MRI	ETI
1	70	F	LSPSS	P	DM	UC	A		N		MET
2	23	F	RSPS	P	NIL	S	A	A	A		TUM
3	56	M	GTCS	P	H,DM,IHD	UC	A	-	N		DRUG
4	48	F	GTCS	P	H	S	A	A	A		CVA
5	28	M	LSPSS	P	NIL	S	A	A	A	A	CVT
6	25	F	GTCS	P	PIH	S	N	N	N		ECL
7	50	M	GTCS	P	PT	S	N	A	A		INF
8	68	M	GTCS	P	HT,IHD	S	A	A	A		CVA
9	56	M	GTCS	P	NIL	S	N	A	A		GRN
10	60	F	LSPSS	P	DM,HT	S	N	N	N		HG
11	72	M	GTCS	P	IHD	S	N	N	N	N	DRUG
12	31	M	GTCS	P	NIL	S	N	N	N		AW
13	50	M	RSPSS	P	PT	S	N	N	A		INF
14	56	M	RSPS	P	DM	S	N	N	N		HG,HYP
15	62	F	RSPS	P	NIL	S	A	N	N		HG
16	45	M	RSPSS	P	NIL	S	N	A	N		ALC
17	35	F	GTCS	P	RHD	S	N	N	N		MET
18	35	M	GTCS	P	NIL	S	N	N	N		AW
19	33	M	GTCS	P	NIL	S	A	N	A		CVA
20	45	M	RSPSS	P	NIL	S	A	N	A		CVA
21	23	F	GTCS	P	PT	S	A		A		INF
22	30	F	RSPSS	P	PT	UC	A	A	A		INF
23	60	F	GTCS	P	HT	S	A	N	A		CVA
24	40	M	GTCS	P	HT	S	A	A	A		ICH
25	37	F	ATON	P	RA	S	N	A	N		CTD
26	34	M	GTCS	P	HIV/PT	S	N	N	N		INF
27	40	F	LSPSS	P	HT	S	A	A	A		CVA
28	62	F	GTCS	P	HT/CAD	S	N	N	N	N	TIA
29	55	M	GTCS	P	HT/CKD	S	N	A	N		MET
30	45	M	RSPSS	P	NIL	S	N	N	N		ALC
31	20	F	GTCS	P	PIH	S	A	N	N		ECL
32	20	F	RSPSS	P	HTDM/RA	S	N	A	A	A	CTD
33	24	F	GTCS	P	SLE,CKD	S	N	A	N		CTD, MET
34	65	M	GTCS	P	HT,	UC	A	-	A		ICH
35	65	F	GTCS	P	HT	UC	A	N	N		MET
36	65	F	EPC	P	DM,HT	UC	A	A	N		MET
37	66	M	LSPSS	P	NIL	S	A	A	A		TUM
38	23	F	GTCS	P	NIL	S	A	A	A	A	CVT,MET
39	34	M	RSPSS	P	HEMO	UC	A	A	A		ICH
40	31	M	GTCS	P	NIL	UC	A	N	N		INF
41	79	M	GTCS	P	DM,HT	S	A	N	A		ICH
42	26	M	GTCS	P	NIL	UC	A	A	N		INF
43	51	M	GTCS	P	DM,HT	UC	A	-	N		MET
44	35	M	GTCS	P	CA LUNG	S	N	N	A		TUM
45	41	M	GTCS	P	NIL	S	N	N	N		ALC
46	47	M	GTCS	P	NIL	S	N	A	A	A	INF
47	36	M	GTCS		NIL	S	N	A	A	A	INF

48	18	M	GTCS	PP	NIL	S	A	N	A	A	INF
49	40	M	GTCS	P	NIL	UC	A	-	N		TOX
50	21	F	GTCS	P	NIL	UC	A	-	N	A	CVT
51	20	F	GTCS	P	NIL	UC	A	-	-		ECL
52	55	F	GTCS	P	NIL	S	N	A	A		TUM
53	23	F	GTCS	P	NIL	S	N	-	N		ECL
54	24	F	GTCS	P	NIL	UC	A	-	N		ECL
55	55	F	GTCS	P	CKD,DM	UC	A	-	N		MET
56	25	F	GTCS	P	NIL	UC	A	A	N		INF
57	36	M	GTCS	P	CAD	UC	A	-	N		HYP
58	25	F	NIL	P	NIL	UC	A	A	A	A	CVT
59	65	M	GTCS	P	HT,DM	UC	A	A	A		CVA
60	37	M	LSPSS	P	DM,PT	UC	A	A	A	A	INF,CVA
61	49	M	LSPSS	P	NIL	S	N	A	N		ALW
62	56	M	GTCS	P	NIL	UC	A	-	N		ALW
63	62	M	GTCS	P	NIL	S	N	N	N		ALW
64	68	F	GTCS	P	NIL	S	N	N	N		ALW
65	74	F	GTCS	P	NIL	S	N	-	N		ALW
66	62	M	LSPSS	P	NIL	UC	A	N	N		ALW
67	54	F	LSPSS	P	HT	S	A	N	N		ALW
68	42	M	GTCS	P	HT,DM	S	A	-	N	A	CVA,MET
69	60	M	GTCS	P	HT,DM	S	A	A	A		CVA
70	51	F	RSPSS	P	NIL	S	A	A	A		CVA
71	59	F	GTCS	P	HT,DM	UC	A	-	A		CVA
72	41	M	RSPSS	P	NIL	S	A	A	A		CVA
73	63	M	GTCS	P	NIL	S	A	A	A		CVA
74	40	F	GTCS	P	NIL	UC	A	A	N	A	CVA,MET
75	58	F	GTCS	P	NIL	UC	A	-	A		CVA
76	53	M	GTCS	P	NIL	UC	A	A	A		CVA
77	48	M	GTCS	P	NIL	S	A	N	A		TUM,MET
78	67	M	GTCS	P	NIL	S	A	A	A		CVA,MET
79	57	M	GTCS	P	NIL	S	A	N	A		CVA
80	64	M	RSPSS	P	NIL	S	A	A	A		CVA
81	54	F	RSPSS	P	NIL	S	N	A	N	A	CVA
82	49	F	GTCS	P	NIL	S	A	A	A		CVA
83	68	M	GTCS	P	NIL	S	A	-	A		CVA
84	70	M	GTCS	P	HT,DM	S	A	-	A		CVA
85	69	M	GTCS	P	HT,DM	S	A	-	A		CVA
86	50	F	GTCS	P	NIL	UC	A	-	A		CVA
87	43	M	GTCS	P	NIL	UC	A	N	N	A	CVT
88	63	F	GTCS	P	NIL	UC	A	A	A	A	CVT
89	79	M	GTCS	P	NIL	S	N	N	A		INF
90	51	F	GTCS	P	NIL	S	A	A	N		INF
91	49	F	LSPSS	P	NIL	S	A	A	N		INF
92	53	F	RSPSS	P	NIL	S	A	A	A		INF
93	69	F	GTCS	P	HT	S	A	A	A		CVA
94	64	M	LSPSS	P	NIL	S	N	A	A	A	GRN
95	71	M	LSPSS	P	NIL	S	N	A	N	A	GRN
96	59	M	RSPSS	P	NIL	S	A	A	N	A	GRN
97	63	F	RSPSS	P	NIL	S	A	N	N	A	GRN
98	53	F	GTCS	P	NIL	S	A	N	A		MET

99	47	F	GTCS	P	NIL	S	N	-	N		MET
100	64	F	GTCS	P	NIL	S	A	N	A		MET
101	54	F	GTCS	P	NIL	S	N	N	A		MET
102	63	M	GTCS	P	NIL	S	A	-	N		MET
103	56	M	GTCS	P	NIL	S	A	A	N		MET
104	70	M	RSPSS	P	NIL	S	A	N	N		MET
105	73	M	GTCS	P	NIL	S	A	N	N		MET
106	63	F	GTCS	P	NIL	S	N	N	N		MET
107	68	F	GTCS	P	NIL	S	A	-	A		TUM
108	69	M	RSPSS	P	NIL	S	A	-	N	A	GRN
109	50	F	GTCS	P	NIL	S	A	-	N	A	GRN
110	56	M	LSPSS	P	NIL	S	A	A	A		TUM
111	43	F	GTCS	P	NIL	S	A	-	A		TUM
112	62	M	GTCS	P	NIL	UC	A	N	A		TUM
113	53	M	GTCS	P	NIL	S	N	A	A		TUM
114	41	M	GTCS	P	NIL	S	N	A	A		TUM
115	63	F	GTCS	P	NIL	UC	A	A	A	A	TUM
116	59	M	GTCS	P	NIL	S	A	A	A	A	TUM
117	66	F	GTCS	P	NIL	S	A	N	A		TUM
118	69	M	GTCS	P	NIL	S	A	-	A	A	TUM
119	37	M	LSPSS	P	DM/HT/TBM	S	A	A	A	A	CVA
120	21	F	RSPSS	P	ITP	S	A	A	A		APLA
121	18	M	LSPSS	P	NIL	S	N	A	A		GRN
122	24	M	LSPSS	P	NIL	S	N	N	A		GRN
123	43	M	RSPSS	P	NIL	S	A	N	A		TUM
124	42	M	GTCS	P	NIL	S	N	A	N	A	AVM
125	42	M	GTCS	P	NIL	S	N	N	N		AW
126	62	M	GTCS	P	PT	S	A	A	A		INF
127	60	M	GTCS	P	NIL	S	N	N	N		AW
128	37	M	GTCS	P	PT	S	N	A	N		INF
129	20	F	GTCS	P	NIL	UC	A		N		HYP
130	36	M	GTCS	P	CAD	UC	A		N		HYP
131	54	F	GTCS	P	HT,DM	UC	A		A		CVA,MET
132	26	M	RSPSS	P	NIL	UC	A	A	N		INF
133	29	M	RSPSS	P	HIV,PT	S	A	A	A		INF
134	45	M	RSPSS	P	RHD	S	A	A	A		CVA
135	20	F	RSPSS	P	SLE	S	A	A	A	A	CTD
136	36	F	RSPSS	P	PT	S	N	A	A	A	INF
137	30	M	GTCS	P	NIL	S	N	A	A		TUM
138	26	F	GTCS	P	ECL	UC	A		A		ECL
139	35	M	GTCS	P	NIL	S	A	A	A	A	CVA
140	32	F	RSPSS	P	NIL	S	N	A	A		INF
141	61	M	GTCS	P	NIL	S	N	A	A		TUM
142	25	M	GTCS	P	NIL	S	A	A	A	A	CVA
143	28	M	GTCS	P	NIL	S	N	N	A	A	AVM
144	26	M	GTCS	P	NIL	S	N	N	A	A	AVM
145	30	F	GTCS	P	SLE	S	N	N	N		CTD
146	51	F	LSPSS	P	NIL	S	N	A	A		TUM
147	31	M	RSPSS	P	PT	S	N	N	A	A	INF
148	75	M	GTCS	P	FIL	UC	A	A	N		INF
149	25	M	GTCS	P	NIL	S	A	N	A		CVA

150	55	M	GTCS	P	NIL	UC	A		A		TUM

KEY TO MASTER CHART

SZT	:	SEIZURE TYPE
HOR	:	HISTORY SUGGESTIVE OF ORGANICITY
SPH	:	SIGNIFICANT PAST MEDICAL HISTORY
HAD	:	HEMODYNAMIC STATUS AT ADMISSION
NEA	:	NEUROLOGICAL EXAMINATION AT ADMISSION
MET	:	METABOLIC ABNORMALITIES AT ADMISSION
CTS	:	CT SCAN
TUM	:	TUMOR
CVA	:	CEREBROVASCULAR ACCIDENT
GRN	:	GRANULOMA
ALW	:	ALCOHOL WITHDRAWAL
INF	:	INFECTIVE ETIOLOGY
CKD	:	CHRONIC KIDNEY DISEASE
CAD	:	CORONARY ARTERY DISEASE
ALC	:	ALCOHOL INTOXICATION
AVM	:	ARTERIOVENOUS MALFORMATION
HYP	:	HYPOXIA
HG	:	HYPOGLYCEMIA
ECL	:	ECLAMPSIA
TOX	:	TOXIC
M	:	MALE
F	:	FEMALE
P	:	PRESENT
GTCS	:	GENERALISED TONIC CLONIC SEIZURES
LSPS	:	LEFT PARTIAL SEIZURES
RSPS	:	RIGHT PARTIAL SEIZURES
S	:	STABLE
UC	:	UNCONSCIOUS
N	:	NORMAL
A	:	ABNORMAL
DM	:	DIABETES MELLITUS
HT	:	HYPERTENSION
CTD	:	CONNECTIVE TISSUE DISORDER